



Peripheral nerve neurostimulation

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Direct electrical stimulation of large-diameter afferent peripheral nerve fibers proximal to the perceived injury site has been used and reported in small published series [1–5] for more than 30 years to treat a variety of intractable painful peripheral mononeuropathy conditions. Originally thought of as a validation of the gate control theory [6,7], the surgical procedure and therapy have been inconsistently applied because of questions regarding appropriate indications, degree of long-term pain control, and surgical techniques. Evolving equipment technologies centered around total implantable systems coupled with newer electrode designs and percutaneous lead implant techniques may herald a renewed interest among pain management specialists to consider peripheral nerve neurostimulation as part of their treatment armamentarium.

For centuries, electricity has been prescribed for a variety of ailments, including gout, face pain, and rather poorly defined “peripheral neuralgias” [8]. Beginning in the mid-1960s, science caught up with empiric observation in the form of strong animal evidence [9] to support the gate control theory of Melzack and Wall, which indicates that stimulation of large-diameter peripheral nerve fibers inhibits small nerve fiber pain transmission [7]. Wall and Sweet [6] (P.D. Wall, personal communication, 2000) stimulated their own infraorbital nerves with percutaneous needle electrodes, producing hypesthesias in the distribution of the targeted nerves. Subsequently, nerve stimulation producing pain relief for an intractable ulnar neuropathy [10] led to early monopolar and bipo-

lar electrode development coupled to radiofrequency-powered electrical sources for peripheral nerve and spinal cord stimulation. By the early and mid-1970s, PNS indications included major peripheral nerve injuries as well as sciatica and even pain from metastatic disease.

Over the past 30 years, electrode design has evolved from hypodermic needle electrical stimulation through nerve-encircling cuff systems to more modern multiarray percutaneous wire electrodes and flat-paddle electrode configurations [11–17]. Power sources have also been transformed from small external battery systems through radiofrequency receiver/external transmitter combinations to total implant lithium ion battery power packs [18–21]. Although PNS implant systems to date are only US Food and Drug Administration (FDA) approved with radiofrequency receivers, most devices today are used as total implant generators in an off-label capacity.

Indications for peripheral nerve neurostimulation

Pain of neurogenic origin rather than from nociceptive generators seems to respond to electrical stimulation to a significant degree [22]. Peripheral nerve trauma and chronic entrapment syndromes, such as failed carpal tunnel or ulnar neuropathy conditions with or without a sympathetic component (CRPS), respond by pain transmission blockade using electrodes implanted proximal to the injury site. Although most upper and lower extremity pain conditions, such as chronic radiculopathy, chronic regional pain syndrome, and even phantom limb and stump neuroma pain, are treated with spinal cord stimulation techniques, identification of a mononeuropathy component not covered by dorsal column

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stimulation may be successfully treated with the addition of a peripherally placed electrode modulated concurrently with the spinal cord stimulation implant.

Patient selection criteria

PNS implant selection criteria should include the following:

- A demonstrated injury for the pain complaint
- Failure of more conservative treatment therapies, including surgery
- No significant drug dependence issues
- Adequate patient motivation and intelligence
- Clear understanding that PNS neuromodulation is designed to help control chronic pain but not to cure the underlying disease process
- Successful trial stimulation
- Identification of the specific injured and painful nerve using selective nerveroot–blocking techniques

Surgical implant techniques

One of the main limiting factors in prescribing and using PNS as a treatment modality has been the requirement for extensive surgical dissection and electrode placement in the region of an at times already injured peripheral nerve. Newer percutaneous electrode placement techniques will allow for more frequent use of PNS in a variety of chronic pain conditions.

Open nerve dissection technique

The most common peripheral nerves treated with PNS include the ulnar, median, radial, posterior tibial, and common peroneal nerves. A flat-paddle electrode array (Resume On-Point Electrode; Medtronic, Minneapolis, MN) has been used most frequently for lead implantation, incorporating a thin mesh apron to facilitate anchoring to the surrounding tissues (Fig. 1).

• Lead implant technique

1. Intravenous sedation and local anesthetics are reasonably well tolerated and will allow for intraoperative stimulation.
2. Dissect and expose a 5-cm segment of peripheral nerve proximal to the injury site completely free from surrounding tissues.

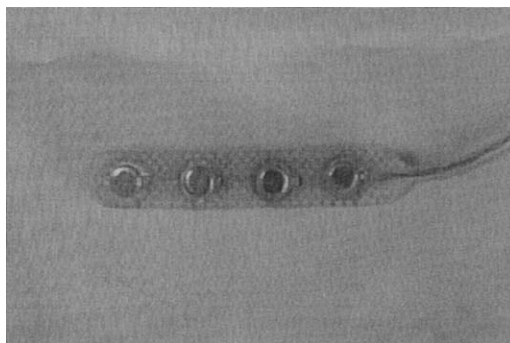


Fig. 1. Resume paddle electrode (Medtronic).

3. Create a flap from nearby fascial tissue or harvest a free fascial graft to place over the electrode, thereby avoiding direct electrode contact with the nerve.
4. Place the electrode lead under the dissected section of nerve longitudinally so that all four electrode contacts remain in close proximity to the nerve (Fig. 2).
5. Secure the electrode to the surrounding muscle fascia with nonabsorbable sutures (Fig. 3).

• Intraoperative screening

1. Temporary stimulation of the electrode array will confirm proper lead position.
2. Externalization of the distal electrode wiring can be done to allow for prolonged postoperative screening stimulation before permanent implantation.
3. Single-stage electrode and power source implantation can also be accomplished via tunneling of an appropriate length of lead extension wire to the receiver/generator pocket.

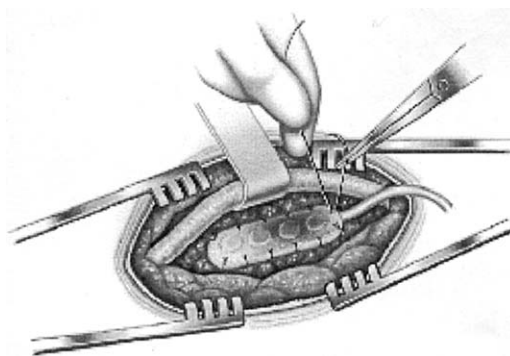


Fig. 2. Electrode placement beneath nerve.

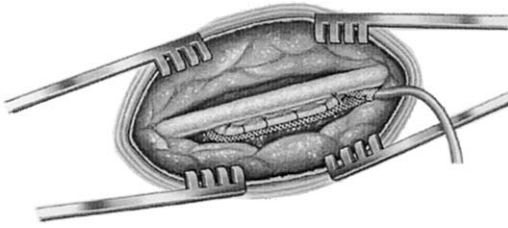


Fig. 3. Fascia-covered electrode in place under major peripheral nerve.

- Receiver/generator placement
 1. Power sources for consideration in PNS implantation are either FDA-approved radiofrequency receiver systems or off-label implantable programmable generators (IPGs). These devices are most commonly placed subcutaneously in the anterior chest or abdominal wall, midaxillary midthoracic region, or posterior superior buttock region (Fig. 4). Lower extremity PNS power sources may be implanted in the lateral thigh or extending into the abdomen (Fig. 5).
- Stimulation parameters
 1. Voltage requirements for PNS are typically much lower than with spinal cord systems with ranges of 0.2 to 3.0 V common. Pulse widths range from 120 to 400 microseconds and frequency of 40

to 100 Hz. Some pain syndromes seem to be frequency dependent for adequate pain relief and require frequencies of 1000 Hz or more, which can only be obtained currently with radiofrequency systems.

Percutaneous electrode implant technique

Extensive experience with percutaneous epidural electrode placement for spinal cord stimulation has been applied to peripheral nerve neuromodulation as well [17]. Simple percutaneous perineural placement of wire electrode arrays parallel to a major peripheral nerve can be accomplished quickly and easily, thereby avoiding extensive nerve dissection surgery. This application has been most effective in failed carpal tunnel syndrome and failed ulnar transposition cases in which the nerve segment in the midforearm or midhumerus, respectively, can be approached at an angle of approximately 20° to 30° with or without nerve conduction/electromyographic (EMG) testing for electrode placement. Anchoring, tunneling, and power source pocketing are the same as with the open dissection technique.

Subcutaneous neurostimulation

Experience with peripheral nerve electrical stimulation for painful mononeuropathies and regional sympathetic dystrophy/chronic regional pain syndrome (RSD/CRPS) syndromes involving

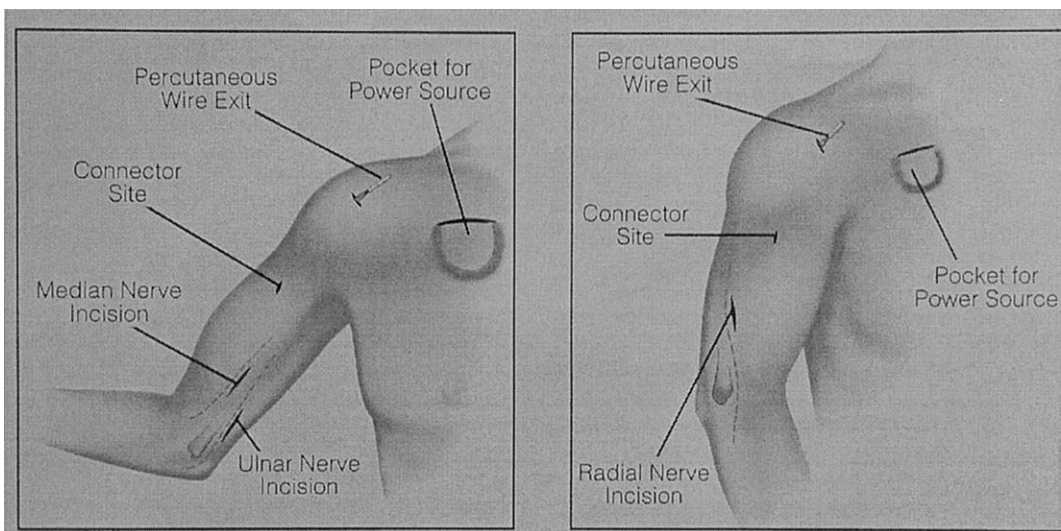


Fig. 4. Upper extremity peripheral nerve neurostimulation placement.

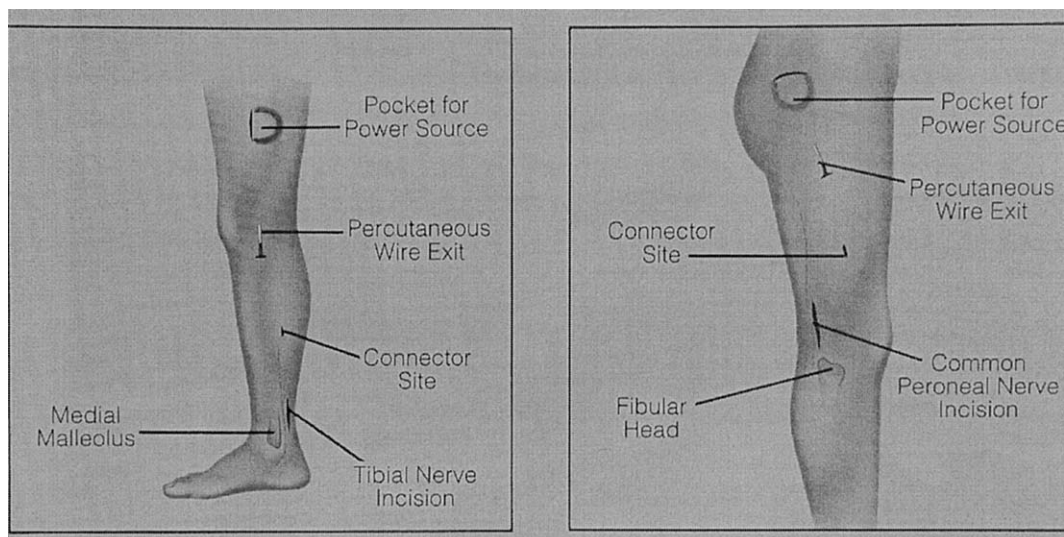


Fig. 5. Lower extremity peripheral nerve neurostimulation placement.

primarily one major nerve led to an important observation that the subcutaneous tissues throughout the body, not necessarily in direct proximity to major peripheral nerves, conduct electrical impulses in a dermatomal and possibly myotomal pattern of paresthesias, producing pain control [20]. This has successfully been applied for the treatment a variety of a medically intractable transformed migraine headaches, occipital neuralgia, and even chronic ilioinguinal pain.

Subcutaneous neurostimulation indications include the following:

- Occipital/transformed migraine headaches
- Cervicogenic pain
- V1 facial pain
- Failed peripheral nerve surgery
- Adjunct to spinal cord stimulation
- Cluneal nerve pain
- Stump/neuroma pain

Peripheral nerve neurostimulation technique for occipital headaches

1. Prescreening includes successful response to local anesthetic blockade of one or more occipital nerves.
2. All patients must undergo outpatient trial screening with a percutaneously placed subcutaneous trial lead (quadripolar electrode) placed transversely one or two finger breadths below the level of maximal occipital tenderness and connected to an external transmitter for a 5- to 7-day trial.
3. Patient position can be supine, lateral, or prone depending on receiver/generator site placement.
4. C-arm fluoroscopy is used to identify the level of C1, where subcutaneous stimulation of a transversely placed electrode usually produces an agreeable paresthesia sensation in the distribution of the affected suboccipital/occipital region. A variety of electrode arrays may be used, including quadripolar, octapolar, elongated quadripolar, and, on occasion, flat-paddle designs.
5. Infiltrating a local anesthetic only at the incision site, a 2- to 3-cm vertical incision centered at the level of C1 can be made either in the midline or lateral to the painful areas.
6. A gently curved Tuhoy needle (bevel down) is inserted subcutaneously through the vertical incision and passed transversely just distal to the maximal painful area. The needle must remain superficial just under the skin. Placement too close to the underlying fascia or within the cervical musculature will produce a motor response or burning sensation rather than paresthesia (Fig. 6).
7. The needle stylet is removed, and the lead array is inserted to the end of the needle, which is then removed from the tissues (Figs. 7 and 8).
8. A report of burning pain or motor response usually indicates that the electrode is too close

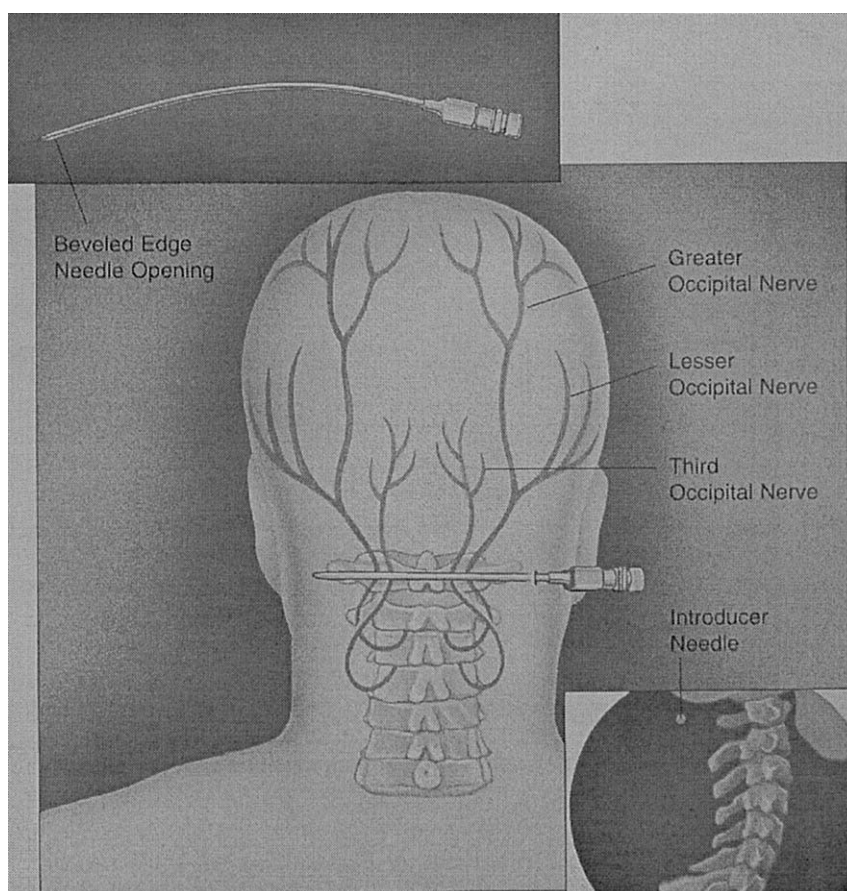


Fig. 6. C1 needle placement for occipital peripheral nerve neurostimulation.

to the fascia or intramuscular and must be repositioned more superficially in the subcutaneous tissue.

9. Anchor the electrode to the underlying fascia, and create a strain-relieving loop (Fig. 9).
10. Tunnel the extension wire, and pocket the receiver/generator to complete the implant.

This procedure, developed and refined since 1992, has achieved excellent long-term pain relief in 55% of patients, good relief in 25% of patients, and fair relief in 15% of patients who have successfully responded to trial stimulation [23].

Peripheral nerve neurostimulation for V1 supraorbital pain

Supraorbital pain from trauma, surgical manipulation, and postherpetic neuralgia has been successfully treated with a subcutaneous implant approach. A quadripolar or octapolar electrode

array is tunneled subcutaneously via a curved Tuhoy needle from behind the frontotemporal hairline, crossing above the supraorbital notch within 1 to 2 cm and producing paresthesias into the surrounding painful region.



Fig. 7. CI curved Tuhoy needle position.



Fig. 8. C1 peripheral nerve neurostimulation electrode position.

The electrode is then anchored to the galea of the scalp and further tunneled posteriorly behind the ear to the receiver/generator pocket in the chest wall or abdomen.

Peripheral nerve neurostimulation for postoperative inguinal pain

Postinguinal herniorrhaphy pain either from nerve entrapment or stretch injury occurs in up to 2% of patients with potential significant functional impairment [24]. Nonoperative neuralgias, including trauma, diabetes, pregnancy, and malignancy, may also be difficult pain management problems. Subcutaneous electrode stimulation after other modality treatment failure has been successful in controlling this condition [25]. Single or twin quadripolar or octapolar percutaneous leads placed subcutaneously in the vicinity of the hernia scar produce paresthesias covering the dermatomal region of the implant with pain relief.

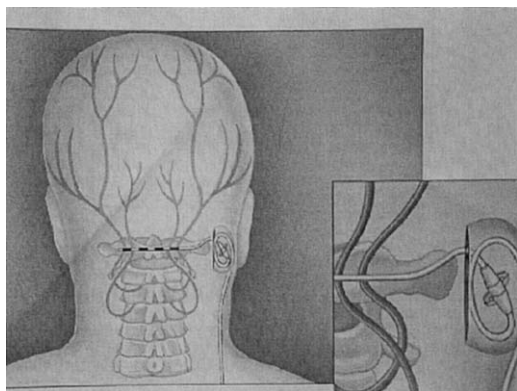


Fig. 9. Loop strain relief.

Complications

There are two main areas of difficulty after wire electrode array implants and flat-paddle electrode placements. Percutaneous wire electrode migration can occur in up to 20% of implants, especially after C1 subcutaneous placement, unless proper anchoring techniques are used. This includes the use of a silicone medical adhesive (Medtronic) suffused between the anchor and electrode with a 22-gauge angiocatheter just before suture tightening to the underlying fascia (Fig. 10).

Additionally, looping the electrode into a pocket adjacent to the incision site will significantly reduce migration potential.

Paddle electrode placement can cause a compression neuropathy to develop if the electrode is not properly anchored, including the possibility of a 90° turn of the paddle into the nerve. Dissection for removal of these electrodes can be difficult secondary to developed scar tissue in and around the nerve/electrode complex.

Mechanism of action

Neurostimulation of peripheral nerve and dorsal spinal cord structures is thought to validate the gate control theory of Melzack and Wall [7], in part, by activation of large-fiber A β fibers producing a C-fiber inhibition at the level of the substantia gelatinosa. Direct blockade of cell membrane depolarization and axonal conduction as well as descending pathway influence from various neurotransmitters, such as serotonin, gamma-aminobutyric acid (GABA), glutamate, and enkephalins, are all influenced by focal elec-

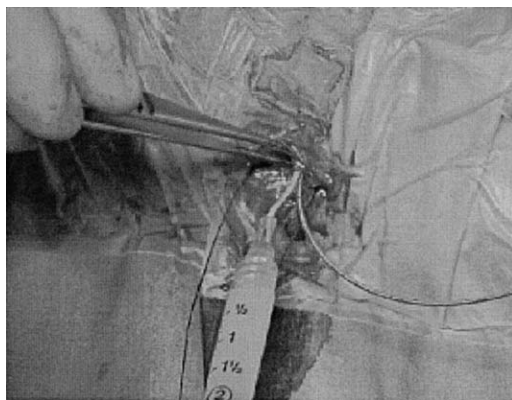


Fig. 10. Medical silicone adhesive injection between anchor and electrode.

trical stimulation for pain transmission modulation [26,27]. The discovery that the subcutaneous tissues conduct electricity in a dermatomal distribution with paresthesia production and subsequent pain relief suggests that alteration in local blood flow, activation of the autonomic nervous system, and antidromic activation of central nervous system structures within the brain stem play a critical role in pain control from neurostimulation. The mechanisms of action for the paresthesia patterns and pain relief obtained from this therapy are incompletely understood but would seem to involve the following elements:

- Subcutaneous electrical conduction
- Dermatomal stimulation
- Myotomal stimulation
- Sympathetic stimulation
- Local blood flow alteration
- PNS
- Peripheral and central neurochemical mechanisms

Recent work with direct electrical stimulation of the greater occipital nerve [28] has shown an increase in metabolic activity in the trigeminal nucleus caudalis and cervical dorsal horn cells in the cat by 220% ipsilateral to the stimulation and by a lesser amount contralaterally. The dorsal horn activity was at the level of C1 and C2, and interaction with the trigeminal innervated structures suggests that the frontally radiating occipital headaches occur as a consequence of overlap of nociceptive information processing at the level of the second-order neurons. Recent positron emission tomography (PET) scan studies in cluster headache patients [21] demonstrate activation of the ipsilateral hypothalamic gray matter region during nitroglycerin spray-induced headaches. These observations suggest the presence of a central trigger mechanism for a variety of headache pain conditions. Peripheral subcutaneous electrical stimulation may influence blood flow within these activated regions via stimulation of the trigeminovascular system at the level of the upper cervical spine.

Outcomes

The long-term success rate (greater than 50% pain relief) for PNS depends on the indication and probably the surgical technique as well. PNS for posttraumatic causalgia/CRPS II has been effective in 60% of advanced intractable cases presenting with symptoms, including allodynia,

vasomotor disorder, trophic changes, motor weakness, and temperature changes [29].

Subcutaneous stimulation for occipital headache syndromes with up to a 9-year follow-up has shown a 70% to 75% success rate, with several distinct subgroups of patients responding to this type of neurostimulation [22]. These include cases of chronic daily transformed migraine headaches that require constant neurostimulation and a group that can successfully abort the onset of a migraine headache by activating their devices during the prodrome of an attack.

Summary

There is a renewed interest in the use of PNS for the control of intractable pain caused by peripheral mononeuropathies and sympathetically mediated chronic pain syndromes. Technical advances in neurostimulation hardware, specifically lead design and surgical advancements with percutaneous and subcutaneous techniques, fuel this interest in part. The use of multipolar electrode arrays placed percutaneously in the region of peripheral nerves or in their dermatomal distribution without the need for extensive surgical dissection should help to support the use of PNS as a reasonable alternative to potentially destructive surgical procedures for chronic pain control.

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